Amendments to the Specification

Please amend the specification as follows:

Please delete the paragraph on page 10, line 3 to line 12 and replace it with the following paragraph:

Figure 1 shows functional presentation of ovalbumin by the transfected tumor cell lines MO4 and EG7. Microcultures were prepared with the T-cell hybridoma RF33.70 (anti-OVA+ K^b) and the indicated number of transfected (squares) or untransfected (circles) tumor cells in the presence (open symbols) or absence (closed symbols) of added exogenous OVA-peptide SIINFEKL **(SEQ ID NO: 1)** (10ng/ml) as described (Rock, et al., 1990, *J. Immunol.* 45:804-811). After 18hrs incubation, supernatants were harvested and assayed for IL-2 using the indicator cell line HT2 (Rock, et al., 1990, *J. Immunol.* 145: 804-811). (A) B16 and the OVA-transfected subclone MO4. (B)-EL4 and the OVA transfected EL4 subclone EG7. OVA presentation by the OVA-transfected tumors was not significantly enhanced by the presence of exogenous SIINFEKL **(SEQ ID NO: 1)** in the assay cultures.

Please delete the paragraph on page 17, line 5 to line 23 and replace it with the following paragraph:

The present invention is exemplified on several fronts using a murine melanoma model described in detail in Example Section 6. Briefly, the primary limitation in studying antigen specific tumor immunity in a murine model is the lack of a defined tumor antigen recognized by MHC class I restricted CTLs. Since TRAs are not fundamentally different from any other protein synthesized by the cell, except that the host is not tolerant to them, a foreign protein synthesized by a tumor should function as a tumor antigen. Tumor immunization methods of the present invention are exemplified with a murine tumor model with a defined, endogenously synthesized TRA by transfecting the ovalbumin (OVA) gene into the C57B1/6 derived melanoma B16. This system is attractive for several reasons: (1) the B16 melanoma is an extensively studied murine tumor, (2) *in vivo* growth characteristics and metastasis of

this tumor line are well characterized, and (3) ovalbumin has a well defined structure. The intracellular processing and presentation of OVA in the C57B1/6 mouse is known. In particular the structure of the processed peptide, presented in association with MHC class I K^b, is known. Assays for the functional expression of ovalbumin peptide SIINFEKL (**SEQ ID NO: 1**) in association with H2-K^b using the T-T hybridoma 33.70.Al anti-OVA-K^b are also known (Kovacovics-Bankowski, et al., 1993, *Proc. Natl, Acad. Sci. USA.* 90: 4942-4946). Techniques to evaluate *in vivo* induction of OVA specific CTLs in this system are also well described (Moore, et al., 1988, Cell 54: 777-785).

Please delete the paragraph on page 23, line 2 to line 11 and replace it with the following paragraph:

The OVA/B16 murine model was used to generate the data disclosed in Example Section 7 and 8 which exemplify the claimed invention. The OVA/B16 murine system is attractive for several reasons: (1) the B16 melanoma is an extensively studied murine tumor, (2) *in vivo* growth characteristics and metastasis of this tumor line are well characterized, and (3) ovalbumin has a well defined structure. The intracellular processing and presentation of OVA in the C57B1/6 mouse is known. In particular the structure of the processed peptide, presented in association with MHC class I Kb, is known. Assays for the functional expression of ovalbumin peptide SIINFEKL (**SEQ ID NO: 1**) in association with H2-Kb using the T-T hybridoma 33.70:Al anti-OVA-Kb are also known (Kovacovics-Bankowski, et al., 1993, *Proc. Natl. Acad. Sci. USA.* 90: 4942-4946).

Please delete the paragraph on page 23, line 24 to page 24, line 2 and replace it with the following paragraph:

After OVA transfection of the B16 melanoma, and selection, the transfected B16 melanoma subclone, MO4 was isolated. The parent melanoma B16, and the OVA transfectant express similar levels of functional Kb on the cell surface as measured by presentation of OVA peptide SIINFEKL (**SEQ ID NO: 1**) to RF33.70

(Figure 1). In contrast, MO4, but not B16, is capable of hybridoma stimulation in the absence of exogenously added peptide (Figure I). This demonstrates endogenous production, processing, and presentation of the transfected antigen. Importantly, endogenous expression of OVA by MO4 did not significantly alter *in vivo* irnmunogenicity of the tumor (Figure 2). Tumor growth (Figure 2A) of B16 and MO4 is comparable in naive mice, as is host survival (Figure 2B).

Please delete the paragraph on page 29, line 25 to page 30, line 5 and replace it with the following paragraph:

In further support of this proposed mechanism is the observation that APCs (bone marrow derived dendritic cells) co-cultured *in vitro* with particulate polynucleotides encoding ovalbumin (prepared as described in Example 7) (pAc-Neo-OVA) can stimulate the OVA SIINFEKL^{+} K^{b} (**SEQ ID NO: 2**) specific T cell hybridoma RF33.70 to produce IL-2 (Figure 4). These data demonstrate that these APCs functionally express the ovalbumin gene and generate the ovalbumin peptide-K^{b} complex. The potency of stimulation is comparable to that observed with APCs pulsed with 2mg/ml of soluble OVA protein. Particulate polynucleotides are effective in this assay using either gold or Fe as the particulate substrate. Thus, the incubation of particulate polynucleotides with phagocytic APCs in vitro results in the endogenous production, processing, and presentation of the transfected antigen. These observations demonstrate that particulate polynucleotides can be taken up and expressed by APCs, and that the corresponding proteins can be functionally presented to induce antigen specific immune responses.